

Decursin Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Decursin

Catalog No.: CFN98509

Cas No.: 5928-25-6

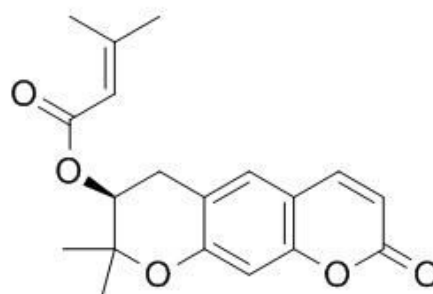
Purity: > 98%

M.F: C₁₉H₂₀O₅

M.W: 328.36

Physical Description: White cryst.

Synonyms: 3-Methyl-2-butenic acid (7S)-7,8-dihydro-8,8-dimethyl-2-oxo-2H,6H
-pyrano[3,2-g]-1-benzopyran-7-yl ester.



[Intended Use]

1. Reference standards;
2. Pharmacological research;
3. Food research;
4. Cosmetic research;
5. Synthetic precursor compounds;
6. Intermediates & Fine Chemicals;
7. Ingredient in supplements, beverages;
8. Others.

[Source]

The roots of *Angelica decursiva*.

[Biological Activity or Inhibitors]

Decursin is a novel inhibitor of NF-kappaB activation in signaling induced by TLR ligands and cytokines, it inhibits induction of inflammatory mediators by blocking nuclear factor-kappaB activation in macrophages.^[1]

Decursin, isolated from the roots of *Angelica gigas*, has anti-tumor activities and exerts its anticancer activity in cells via inhibition of the Wnt/pathway.^[2,3]

Decursin and decursinol angelate inhibited vascular endothelial growth factor (VEGF)-induced phosphorylation of VEGFR-2, extracellular signal-regulated kinases and c-Jun N-terminal kinase mitogen-activated protein kinases, demonstrates that decursin and decursinol angelate are novel candidates for inhibition of VEGF-induced angiogenesis.^[4]

Decursin displays toxic activity against various human cancer cell lines, for which the ED50 of decursin was about 5-16 micrograms/ml; it displays relatively low cytotoxicity against normal fibroblasts; it also activates protein kinase C (PKC) in vitro, which indicates that the cytotoxic activity of decursin may be related to the protein kinase C activation.^[5]

Decursin can block CCl₄-induced liver fibrosis and inhibited TGF-β1-mediated HSC activation in vitro, demonstrates that decursin exhibits hepatoprotective effects on experimental fibrosis, potentially by inhibiting the TGF-β1 induced NOX activation and Smad signaling.^[6]

Decursin and decursinol angelate inhibit estrogen-stimulated and estrogen-independent growth and survival of breast cancer cells, the data provide mechanistic rationales for validating the chemopreventive and therapeutic efficacy of decursin and its derivatives in preclinical animal models of breast cancer.^[7]

[Solvent]

Chloroform, Dichloromethane, Ethyl Acetate, DMSO, Acetone, etc.

[HPLC Method]^[8]

Mobile phase: Phosphate buffer- Acetonitrile- Sodium lauryl sulfate, gradient elution;

Flow rate: 1.0 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 230 nm.

[Storage]

2-8°C, Protected from air and light, refrigerate or freeze.

[References]

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- [2] Lee S, Lee Y S, Sang H J, *et al. Arch. Pharm. Res.*, 2003, 26(9):727-30.
- [3] Song G Y, Lee J H, Cho M, *et al. Mol. Pharmacol.*, 2007, 72(6):1599-606.
- [4] Myunghwan J, Sunhee L, Eunmi A, *et al. Carcinogenesis*, 2009, 30(4):655-61.
- [5] Ahn K S, Sim W S, Kim I H. *Planta Med.*, 1996, 62(1):7-9.
- [6] Choi Y J, Da H K, Sang J K, *et al. Life Sci.*, 2014, 108(2):94-103.
- [7] Jiang C, Guo J, Wang Z, *et al. Breast Cancer Research Bcr*, 2007, 9(6):1-12.
- [8] Kang Y G, Lee J H, Chae H J, *et al. Korean Journal of Pharmacognosy*, 2003, 34(3):201-5.

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